

Supplementary figure legend

Supplementary Fig. 1

The localization of Sgo1-GFP to pre-anaphase spindles is abolished in cells lacking the kinase domain of Bub1. An Spc29-RFP fusion construct was used to visualize the spindle poles. The series of images in the lower panel were captured by live-cell imaging and show the spindle recruitment and subsequent removal of Sgo1-GFP over the course of a single mitosis.

Supplementary Fig. 2

A. *IPL1* over-expression cannot rescue the growth defect of *sgo1Δ* or *bub1Δ* tetraploids. **B.** Alleles of *BIR1* that code for a mutant protein unable to interact with Sli15 (*BIR1*⁹⁰¹) or unable to interact with both Sli15 and Ip11 (*BIR1*^{931,935}) cannot suppress the growth defect of tetraploids lacking either *BUB1* or *SGO1*.

Supplementary Fig. 3

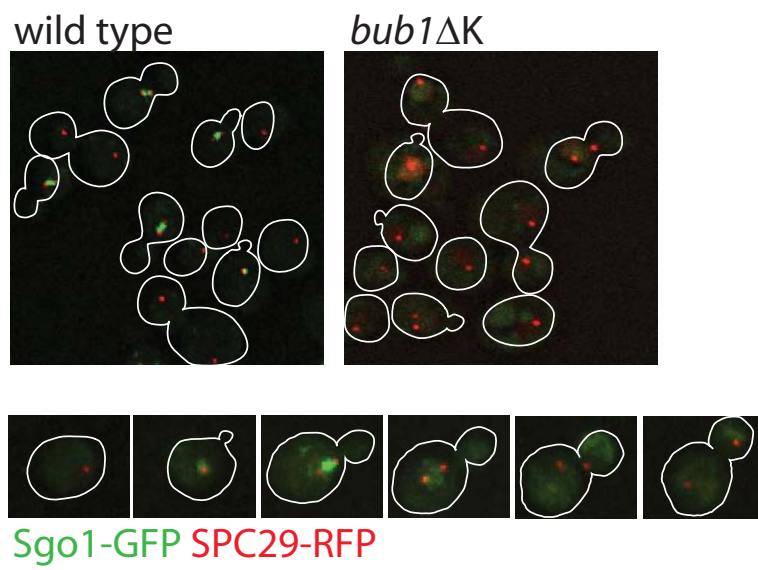
A. Tetraploid cells lacking *SGO1* do not exhibit defects in sister-chromatid cohesion during mitosis, in agreement with previous results in haploid cells. We used tetraploid cells expressing TetR-GFP and marked a single centromere by introducing a TetO array at chromosome IV. Cells were arrested in G1 and in metaphase (M) and scored for the number of distinct GFP foci. **B.** A mutant Sgo1 protein containing isoleucine instead of arginine in position 51 no longer interacts with the phosphatase PP2A, but it can still partially restore the viability of the *sgo1Δ* tetraploids.

Supplementary Fig. 4

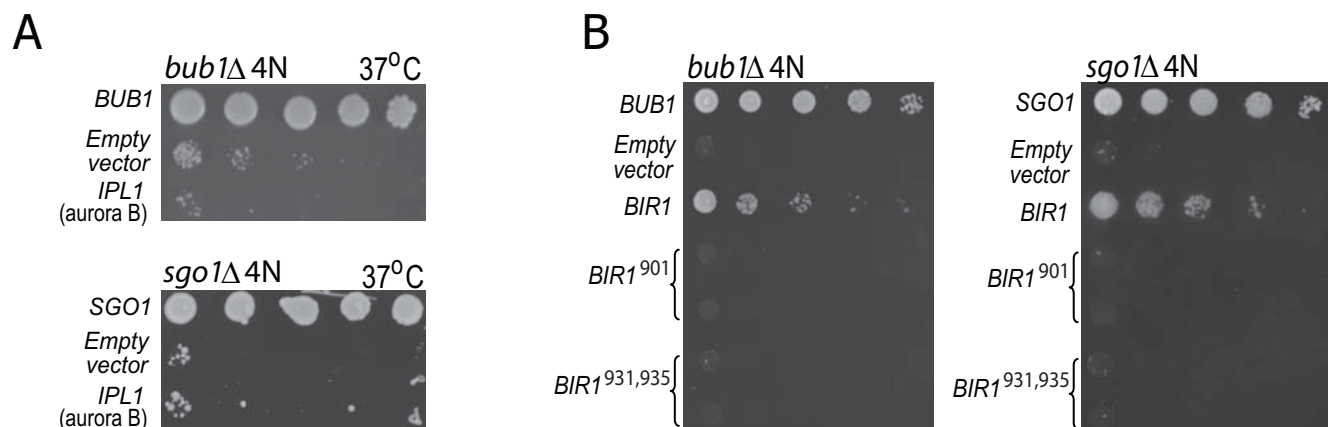
A. The benomyl-sensitivity of cells lacking Cdc55 (a regulatory subunit of the phosphatase PP2A) cannot be suppressed by the over-expression of *BIR1* or *SLI15*. **B.** Cdc55 is not required for the ability of over-expressed *SGO1* to suppress the benomyl sensitivity of haploid *bub1Δ* cells.

Supplementary Fig. 5

A. Over-expression of *MPS1* on a 2-micron plasmid partially rescues the growth defect of *sgo1Δ* tetraploids. **B.** Over-expression of *MPS1* on a 2-micron plasmid partially rescues the sensitivity of *sgo1Δ* haploid to benomyl. **C.** Over-expression of *MPS1* rescues the benomyl sensitivity of the checkpoint-competent haploid strain *bub1ΔK* (lacking the kinase domain) but not the checkpoint-deficient strain *bub1Δ*.

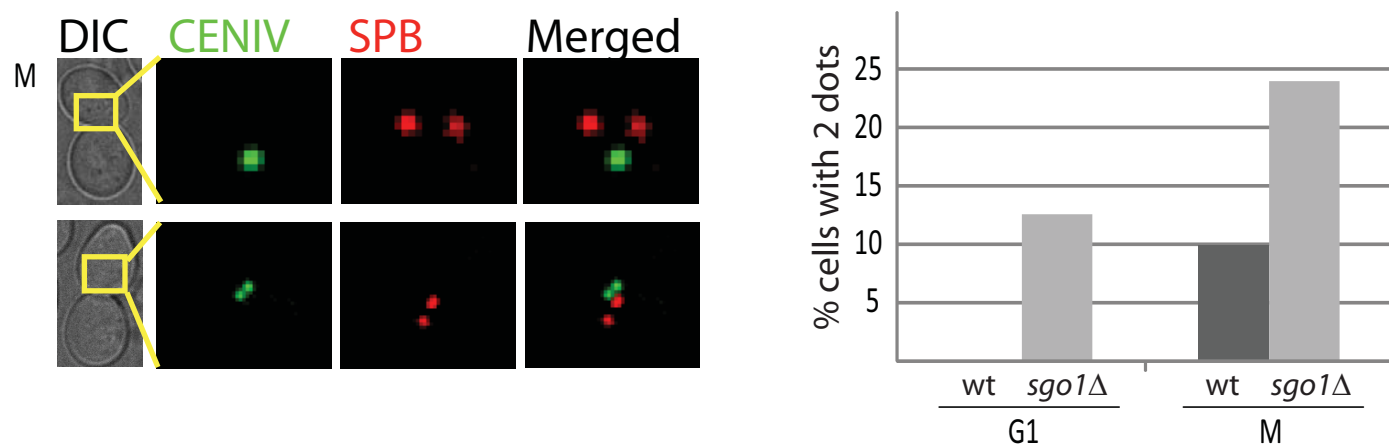


Supplementary Figure 1, Storchova, Becker et al.

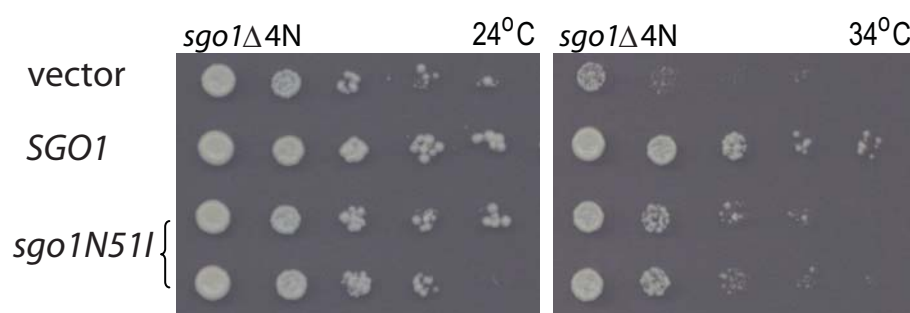


Supplementary Figure 2, Storchova, Becker et al.

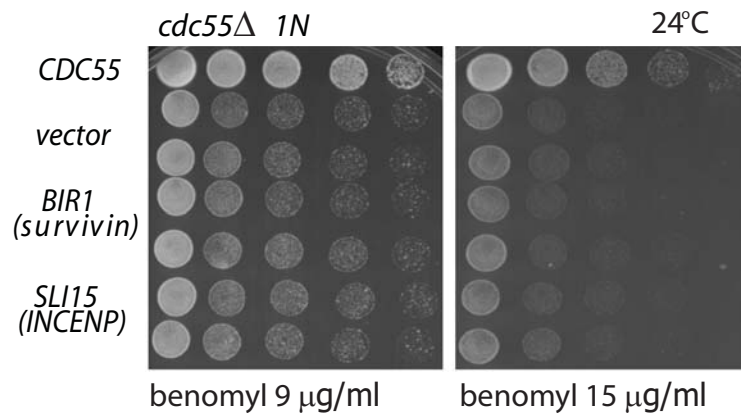
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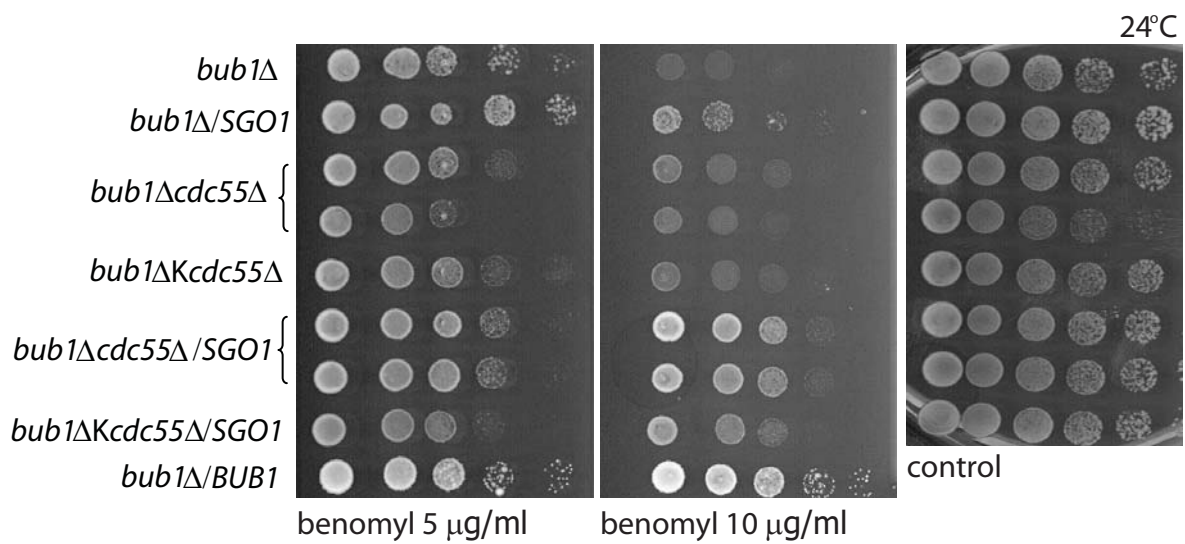
B



A

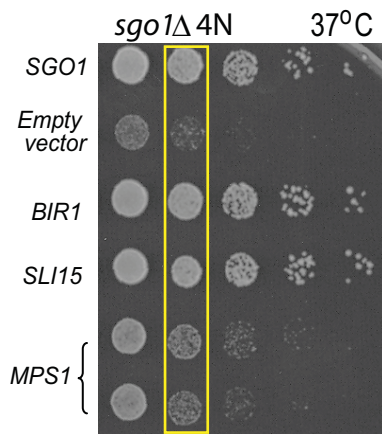


B

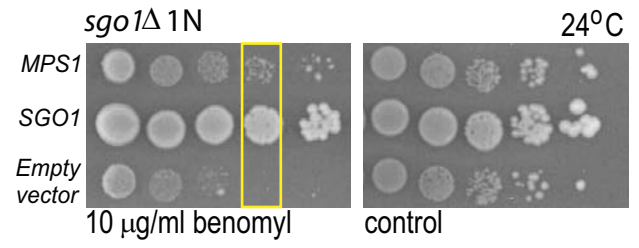


Supplementary Figure 4, Storchova, Becker et al.

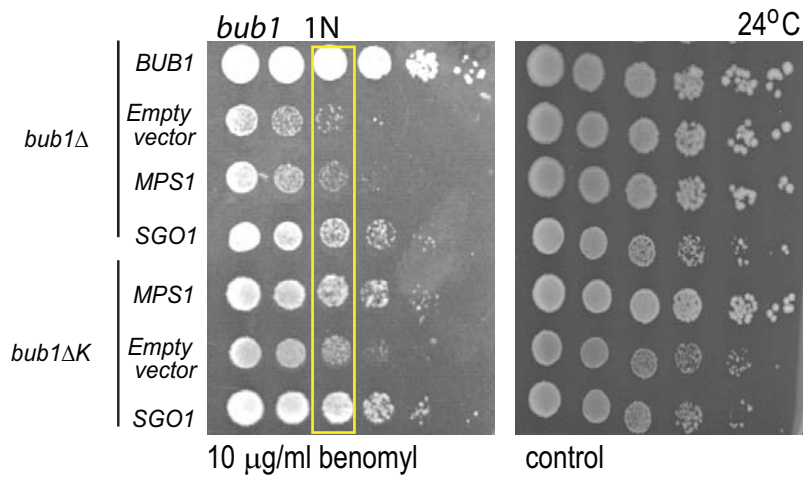
A



B



C



Supplementary Figure 5, Storchova, Becker et al.